# *Modern Concepts of Cardiovascular Disease*

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## HEART DISEASE IN RELATION TO BRONCHIAL ASTHMA AND EMPHYSEMA

Until recently, it was believed that chronic bronchial asthma as well as emphysema, which is its constant sequel, resulted sooner or later in heart failure. The mechanism of cardiac damage seemed quite obvious, for the pulmonary capillaries carrying blood from the right side of the heart to the left were found to be narrowed and broken in the greatly distended lungs examined at autopsy. To drive blood through the reduced capillaries in such lungs might well throw a strain on the right ventricle, which would in time hypertrophy, dilate and eventually fail. This supposition had clinical support, for obvious signs of cardiac decompensation were found commonly in elderly individuals with the rounded "barrel" chests typical of senile emphysema. In younger patients, too, who had had asthma for a long time, the findings of shortness of breath on exertion as well as cyanosis, each occurring between paroxysms, seemed to indicate some trouble in the heart.

Quite recently both clinical and pathological evidence have been presented which throw considerable doubt upon the theory that heart failure is the natural consequence of asthma and emphysema. In the past few years several investigations of the heart in cases of long-standing asthma have been made. Almost invariably emphysema was present. Some of these studies were very thorough. Modern diagnostic methods were used and critical interpretation of the findings made. There was general agreement that in the great majority of the patients examined no direct evidence of heart damage was found.

Soon after these reports were published, attention was directed to the condition of the heart as it appeared at autopsy in cases of asthma and emphysema. It then became apparent how very infrequently such deaths occurred. About ten years ago patients with bronchial asthma who had been reported in recent medical literature as coming to postmortem examination numbered about twenty. Since that time perhaps another thirty have been added. In the great majority of these instances, the heart showed no pronounced lesion. The infrequency of death from asthma implies that if heart disease should be common among patients, it would be of no great severity.

Similar studies were undertaken to determine if emphysema in the absence of asthma likewise would leave no imprint on the heart. Until recently no deliberate examination of this question had been made. Scattered reports in the literature were confusing. Investigation proved that unlike bronchial asthma, many cases of emphysema showed diseased hearts at postmortem. It is noteworthy, however, that most of these instances were in elderly people with senile emphysema. When the emphysema resulted from bronchial obstruction, the heart was usually normal.

It is now recognized that senile emphysema is a condition very different from the type following bronchial obstruction. As a matter of fact, in many cases it is not emphysema at all in the sense that the lung has lost its elasticity, or that alveoli are stretched and broken. The underlying lesion is not

in the lungs but in the spine, which is deformed. This results in a barrel shaped chest which causes the lungs to alter their normal position.

When senile emphysema is excluded from statistical studies of the heart at autopsy, and only cases of obstructive emphysema are considered, heart lesions are uncommon. When they do occur they usually appear as a generalized hypertrophy which involves both the right and left ventricles. No adequate explanation is at hand for this phenomenon. Very rarely the right ventricle alone is enlarged, but only in cases where emphysema is associated with arteriosclerosis of the pulmonary artery.

The fact that there is little clinical or pathological evidence of heart disease in chronic asthma and emphysema does not appear in keeping with the older observations that the capillaries of distended lungs are narrow and broken. Recent physiological studies have shown, however, that there is a great reserve in the pulmonary circulation. One must obliterate over one-half of the pulmonary capillary bed before there is any effect on the right heart. As a matter of fact, it has been demonstrated that as the lungs distend the capillaries straighten and fluid passes through them more readily than when they are not inflated. The observation that in chronic asthmatics, as well as in patients with obstructive emphysema, dyspnea on exertion and cyanosis are common does not necessarily imply heart disease. These signs can be entirely accounted for by faulty ventilation in the lungs.

In obstructive emphysema, the blood pressure is not elevated and may be below average values.

It is evident, therefore, that contrary to older theories, neither asthma nor emphysema cause any material damage to the heart in the majority of cases. When lesions do occur, they are in the heart muscle and not on the valves. There is first generalized enlargement, and in time increase of cyanosis and shortness of breath on exertion appear. Dependent edema, orthopnea, râles at the bases of the lungs, and other usual signs of decompensation follow. In the earlier stages, these indications of heart damage are difficult to detect by physical signs alone. The cardiac enlargement is obscured on percussion by the hyperresonant lungs, which overlap the heart and muffle the sounds. X-ray or fluoroscopic examination of the chest, however, will readily distinguish cases in which the heart is involved. The cardiac diameters are increased in contrast to the typical elongated or spindle-shaped heart of emphysema. In the latter instance the heart is stretched lengthwise by its attachment to the diaphragm, which is held descended by the voluminous lungs.

The treatment of cardiac decompensation from emphysema is similar to that from other causes. Rest and digitalis are the two important principles. It is interesting that rest is very beneficial in most cases of obstructive emphysema, for the underlying bronchitis is thereby benefited and bronchial obstruction becomes less. Digitalis, as well as rest, has been given to many such patients with alleged benefit, and this apparent therapeutic test has served to maintain the belief that the heart is usually affected. Rest, however, without digitalis will secure the same result in most cases, unless true decompensation occurs.

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## SELECTED ABSTRACT

Camp, P. D. and White, P. D.; Pericardial Effusion—A Clinical Study. Amer. Jour. Med. Sci. 184:782: (Dec.) 1932.

These authors in a careful analysis of 1729 autopsies at the Massachusetts General Hospital found that pericardial effusion of more than 100 cc. was present in 126 instances (*7.2%*). This fact stresses the fairly frequent presence of pericardial effusion in small amounts (94 had less than 250 cc.). A correlation of autopsy and clinical data indicated that except when acute fibrinous pericarditis occurs, the diagnosis of pericardial fluid is not apt to be made if less than 500 cc. of fluid is present, even when X-rays are taken. In this series only four cases were correctly diagnosed before death out of 118 showing an effusion of less than 500 cc. The X-ray, however, can sometimes detect small amounts of fluid that cannot be recognized clinically.

M.N.F.